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Published in:
Journal of Dairy Research

DOI:
10.1017/S0022029915000059

First published: 04/02/2015

Document Version
Peer reviewed version

Association of lameness with milk yield and lactation curves in Chios dairy ewes

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Lameness and milk yield in dairy ewes

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SUMMARY

The objective of the study was twofold: (i) to quantify the differences in daily milk yield (DMY) and total milk yield (TMY) between lame and non-lame dairy ewes and (ii) to determine the shape of lactation curves around the lameness incident. The overall study was a prospective study of lameness for the surveyed sheep population, with a nested study including the selection of matching controls for each lame ewe separately. Two intensively reared flocks of purebred Chios ewes and a total of 283

ewes were used. Data, including gait assessment and DMY records, were collected on a weekly basis during on-farm visits across the milking period. A general linear model was developed for the calculation of lactation curves of lame and non-lame ewes, whereas one-way ANOVAs were used for the comparisons between lame ewes and their controls. Lameness incidence was 12.4% and 16.8% in Farm A and B, respectively. Average DMY in lame ewes was significantly lower (213.8 g, $P<0.001$) compared with the rest of the flock, where DMY averaged at 1.340 g. The highest DMY reduction in lame ewes was observed during the 16th week of the milking period ($P<0.001$), whereas, the reduction of DMY, for lame ewes, remained significant at $P<0.001$ level from the eighth to the 28th week of milking. The comparisons between lame and controls revealed that at the week of lameness diagnosis a significant DMY reduction ($P\leq0.001$) was observed in lame ewes (about 32.5%), which was maximized one week later (35.8%, $P\leq0.001$) and continued for several weeks after recovery, resulting in 19.3% lower TMY for lame ewes for the first 210 days of milking period ($P<0.01$). Moreover, at flock level, TMYs for non-lame and lame ewes, as calculated by the general linear model, were 318.9 kg and 268.0 kg, respectively. The results of this study demonstrate an evidence of significant financial losses in dairy sheep due to lameness, which though, need to be accurately estimated in further, more detailed, analyses.

KEYWORDS: lameness, dairy sheep, milk yield, lactation curves

INTRODUCTION

Lameness is a departure from normal gait, caused by disease or injury in some part of limbs or trunk, usually accompanied with pain (Boden, 1998). The aetiology can be broadly classified as either genetic, congenital, physical injury or infection (Coulon *et al.* 1996; Green *et al.* 2002; Winter, 2004). The notion is that lameness is one of the most important health problems in sheep, related not only to impaired animal welfare but to production losses, as well. Most of the available information on sheep lameness relates to meat/wool producing breeds, with well documented evidence of the causes, prevalence, incidence and economic consequences (Green & George, 2008; Kaler & Green, 2008), which include weight loss, reproductive failure and reduced wool production (Stewart *et al.* 1984; Marshall *et al.* 1991; Eze, 2002). However, it is dairy sheep production that is the major industry in Greece and most Mediterranean countries (De Rancourt *et al.* 2006; Gelasakis *et al.* 2012), with its renowned culinary specialties, like Feta and Roquefort cheeses. Therefore, detailed information regarding the effect of lameness on sheep milk production is warranted.

In dairy sheep, lameness incidence has been found to show high variability depending on both physiological and environmental factors (Gelasakis *et al.* 2013). Moreover, in the majority of cases and irrespective of the problem's magnitude within the flocks, farmers underestimate lameness incidence and tend to disregard the negative effects of lameness on milk production (Gelasakis *et al.* 2010). This attitude bears a striking resemblance with that of dairy cow farmers (Espejo *et al.* 2006; Leach *et al.* 2010). It is well established, though, that lameness is associated with a significant reduction in milk yield in this species (Warnick *et al.* 2001; Green *et al.* 2002; Bicalho *et al.* 2008). Further research is expected to facilitate the better

understanding of the significance of the problem in dairy sheep as in the case of dairy cows (Huxley, 2013).

Besides the welfare issues, one factor that could raise dairy sheep farmers' awareness on lameness is to demonstrate its cost. In this respect, the quantification of lameness impact on milk production is a prerequisite. Moreover, as with all diseases, early detection is crucial for timely intervention and successful treatment; visual identification (locomotion scoring) of lame ewes is a subjective, time consuming and difficult method to apply (Kaler *et al.* 2009, Phythian *et al.* 2013) considering the natural tendency of most sheep to congregate at the sight of humans observing or approaching them. An objective variable would be very useful, especially if it could alert farmers early in the course of the disease.

Hence, the objective of the present study was twofold. First, to quantify the differences in daily milk yield (DMY) and total milk yield (TMY) between lame and non-lame ewes and secondly, to determine the shape of lactation curves around the lameness incident in order to explore the possibility to use milk recording data as an early diagnostic tool.

MATERIALS AND METHODS

Two intensively reared flocks of purebred Chios ewes were used for the study. Flock monitoring and data collection pertained to the period 2008-2009. The study has been approved by the ethics review committee of the School of Veterinary Medicine, Aristotle University of Thessaloniki.

Animals and management

A total of 170 and 113 ewes that lambed from October through December 2008, on Farms A and B, respectively, were considered for the study. Both farms were located in Northern Greece (Farm A: 20m above sea level, latitude 40˚17'18'', longitude 23˚09'29'' and Farm B 107 m above sea level, latitude 39˚22'43'', longitude 22˚51'37''). A sheep shed providing a floor area of 2 m^2/ewe and a volume of about 10 m^3/ewe was available on Farm A, but ventilation was moderate. On Farm B, a shed providing a floor area of 2 m^2/ewe and a volume of 12 m^3/ewe was available while ventilation was adequate in this case; fans were installed and operated when necessary. Barley straw was used as bedding on both farms. During winter, fresh bedding was added every other day; in spring and summer periods this interval was extended to 5-10 days, depending on bedding condition. The bedding was removed and premises were disinfected twice a year on Farm A and three times per year on Farm B, using a combination of commercial disinfectants and lime. Ewes had access, year round, to an earthen exercise paddock (2.5 m^2/ewe).

On Farm A lambing started at the end of October and peaked in late November. Lambs were kept with their dams for about eight weeks. On Farm B, oestrus synchronization with intravaginal sponges resulted in a short lambing period of about 10 days, in mid November. Lambs were artificially reared for eight weeks.

Ewes were machine-milked three times per day for three months and thereafter, twice a day until the end of the milking period, which lasted about eight months. Milking parlours were equipped with automatic milk recording systems for individual ewes (SAE Afikim – Afimilk and Alpro - De Laval, for Farms A and B, respectively).

On Farm A, feeding of ewes during the experimental period was based on alfalfa hay (1.0-1.6 kg/ewe/day), barley straw (0.2-0.5 kg/ewe/day) and concentrates (0.7-1.5 kg/ewe/day) comprising of corn grain (35.0%), barley grain (32.5%), soybean meal (30.0%) and a mineral/vitamin supplement (2.5%). The amount of ration offered was adjusted to group milk yield and pasture availability. Rations were offered in troughs allowing sufficient space (0.3 m/ewe), to enable access of all ewes at the same time. A five-hectare sown irrigated pasture (*Lolium perenne* + *Trifolium repens*) was available for grazing from March until September. On Farm B, feeding of ewes was based on alfalfa hay (0.8-1.4 kg/ewe/day), barley straw (0.1-0.4 kg/ewe/day), corn silage (1.0-2.0 kg/ewe/day) and concentrates (0.7-1.3 kg/ewe/day) comprising of corn grain (37.0%), barley grain (23.0%), soybean meal (16.0%), wheat bran (10.0%), sunflower cake (10.0%) and a mineral/vitamin supplement (4.0%). The amount of ration offered was adjusted to group milk yield. Rations were offered on a feeding belt (0.33 m/ewe) which enabled access of all ewes at the same time.

A well-designed vaccination protocol against Brucellosis (*Brucella melitensis* vaccine, strain Rev. 1), Clostridial diseases (Covexin 8A; Schering-Plough Animal Health), Contagious agalactia (Agalax; CEVA), Chlamydial abortion (Enzovax; Intervet International B.V.) and Paratuberculosis (Gudair Vaccine; Provet) was strictly followed in both flocks. Regarding parasites, ewes were treated with ivermectin (0.2 mg/kg Valaneq; Merial) and fenbendazole (Farm A, 10 mg/kg Panacur; Intervet) or netobimin (Farm B, 10 mg/kg Hapadex; Schering-Plough Animal Health) at the third month of gestation and at lambing, respectively. All ewes were treated with an intramammary antibiotic preparation (Nafpenzal Dry Cow; Intervet International) at dry-off (extra-label use). Routine foot trimming was carried out once a year, at lambing. After the diagnosis of lameness, lame ewes were treated

using a single intramuscular injection of long acting Oxytetracycline (Alamycin LA; Norbrook) at a dose rate of 20 mg/kg.

Experimental design

The overall study was a prospective study of lameness for the surveyed sheep population. For the implementation of the study, the same veterinarian visited the farms once a week throughout the entire milking period resulting in a total of 34 visits per flock. Milk yield was electronically recorded daily for individual ewes in both flocks. For the subsequent statistical analyses seven-day average milk yields were used representing the average DMYs for the week of visit. Average DMYs, also, enabled the calculation of lactation curves and enabled the comparisons between lame ewes and the selected controls regarding milk yield for the pre- and postlameness period.

Ewes were observed twice daily (in the milking parlour) by the farm owners or the personnel for any abnormal behaviour. On both farms, a passageway that allowed ewes to enter the milking parlour in single line was constructed to allow gait observation of individual ewes. Ewes showing signs of disease or a sudden reduction in DMY were clinically examined by the veterinarian at the next visit. When a ewe was found lame, then a healthy one of the same age, same number and stage of lactation, similar milk potential (previous lactation records) and average DMY at the beginning of current milking period was chosen as a control. The selection was based on data from the farm's electronic records. Both animals were colour-marked to help identify them after milking for further testing. Clinical examination, microbiological examination of milk samples and parasitological examination of faeces were performed both on lame and control ewes in order to identify and exclude from the

168 study ewes either showing clinical signs of diseases or with subclinical mastitis and/or
169 high levels of parasitic infestation. The examinations and tests performed are
170 summarized below:

171 **(i) Clinical examination:** It comprised inspection (head, body, limbs, feet and
172 conjunctivae), palpation of udder and joints, as well as auscultation of lungs and heart.
173 Heart rate, breathing rate and body temperature were recorded. Also, body condition
174 score (BCS) was assessed using the five-point scale, from 1 (emaciated) to 5 (obese),
175 proposed by Russel *et al.* (1969).

176 **(ii) Locomotion Score (LS) and lameness:** Locomotion assessment was based on the
177 following four-point scale scoring system (Hill *et al.* 1997): 1= Normal gait, 2= No
178 obvious lameness when standing, abnormal gait when walking, 3= Shifting stance and
179 obvious lameness when walking, 4= Unwilling to bear weight on one foot when
180 standing or walking. Ewes with a locomotion score higher than 1 at least once
181 throughout the milking period were considered to be lame. All other ewes were
182 considered non-lame for the purposes of this study. The cause of lameness was
183 assessed during the clinical examination by an experienced veterinarian. Lame feet
184 were inspected through observation and palpation in order to localize possible
185 abnormalities, injuries, lesions or painful sites. Afterwards, a detailed foot-trimming
186 was performed in order to reveal any lesions underneath the hoof wall; final diagnosis
187 of foot lameness was set on the basis of the lesions and the clinical manifestation of
188 the hoof disease.

189 **(iii) Milk sampling and assessment:** Milk samples were taken for California Mastitis
190 Test (CMT, Bovi-Vet; Kruuse) and bacteriological examination, to test for subclinical
191 mastitis (Fthenakis *et al.* 1991).

(iv) Parasitological examination: Faecal samples were collected directly from the rectum and were examined for faecal egg counts (FECs) using the modified McMaster method (Ministry of Agriculture, Fisheries and Food, 1986).

Examination and testing of case and control animals continued throughout the milking period. On Farm A, 21 out of 170 ewes were found lame due to foot lesions; four of them were excluded from the analysis due to subclinical or clinical mastitis of either the lame or the control ewe, at some point of the study. On Farm B, lameness was diagnosed in 19 out of 113 ewes. Seven of them were excluded from analysis due to health problems (subclinical mastitis, metritis, hernia) or insufficient data. Finally, 17 and 12 lame ewes from Farms A and B, respectively, were used in the subsequent statistical analysis.

Data management and statistical analysis

(i) Descriptive statistics

Initially, descriptive statistics were calculated including means and standard errors of means for DMY and for TMY of the first 210 days of milking period, of lame ewes and their selected controls.

(ii) Lactation curve calculation

A general linear model was developed for the calculation of lactation curves of lame and non-lame ewes across milking period using ASReml (Model 1). In each flock, the first lameness event during milking period was used for each ewe.

$$\mathbf{TDM_{abcdkghj} = m + F_a + YM_b + LA_c + MY_d + W_k + E_gW_k + L_h + S_j + e_{abcdkghj}}$$

(Model 1)

Where:

215 $TDM_{abcdkghj}$ = average DMY for the g^{th} ewe of the a^{th} flock measure on the k^{th} week of
216 milking period (kg),

217 m = overall mean,

218 F_a = fixed effect of the a^{th} flock (2 levels),

219 YM_b = fixed effect of the b^{th} interaction between lambing year and lambing month,

220 LA_c = fixed effect of the c^{th} interaction between the number of lactation and age at
221 lambing (in months),

222 MY_d = fixed effect of the d^{th} interaction between the month and the year DMY was
223 calculated,

224 W_k = fixed effect of the k^{th} week of milking period when DMY was assessed (a
225 second order polynomial was used in order milk yield curves and covariances for
226 repeated measures of the same ewe to be considered),

227 E_gW_k = random effect of the interaction between the g^{th} ewe and the k^{th} week of
228 milking period when DMY was assessed (a second order polynomial was, also, used
229 for the same reasons described above),

230 L_h = fixed effect of the h^{th} lameness status (2 levels, 1= non-lame ewes, 2= lame
231 ewes),

232 S_j = fixed effect of the j^{th} week postlambing,

233 $e_{abcdkghj}$ = random residual.

234 DMYs of lame ewes and their selected controls (adjusted for number and
235 week of lactation) were compared using one-way analysis of variance (one-way

ANOVA); comparisons were performed per week for the period initiated four weeks before lameness onset and were completed eight weeks after it.

Effect of lameness on TMY

TMY was calculated for all ewes based on the average weekly DMY solutions produced by model 1. Moreover, one way ANOVAs were used in order to compare TMY between lame ewes and their controls.

RESULTS

Lameness incidence on Farms A and B was 12.4% and 16.8%, respectively. The majority of lameness cases were diagnosed during the first four months of lactation both on Farm A (82.4%) and B (66.7%). Aetiology and duration of lameness are presented in Table 1. White line abscesses (WLA) were the major causes of lameness (70.6% and 58.3% of cases on Farms A and B, respectively) followed by footrot, pedal joint abscesses (PJA) and injuries. Locomotion score was equal to 2 for most of the WLA cases (66.7%) on Farm A and footrot and PJA were associated with severe lameness (LS=4). On Farm B, the majority of cases were assigned a locomotion score equal to 3, regardless the cause of lameness. Duration of lameness was longer than a week in 83.3% and 47.1% of cases on Farms A and B, respectively (Table 1) (Table 1 near here). In the same table, it is obvious that irrespective of the etiology, most of the lameness cases occurred from January to April. In particular, white line lesions were most prevalent in January and February, whereas, all of the footrot cases were observed between February and April. At the end of the lactation period (in July) no cases of lameness were observed.

All factors fitted in Model 1, including lameness, had a significant effect on DMY ($P<0.05$). Average DMY in lame ewes was significantly lower (213.8 g, $P<0.001$) compared with the rest of the flock, where DMY averaged at 1.340 g (a reduction of about 16%). Figure 1 shows the DMY curves for non-lame and lame ewes across the milking period. Mean DMY was 1.89±0.107 kg and at 1.86±0.061 in the beginning of the milking period for lame and non-lame ewes, respectively ($P>0.05$). Afterwards, DMY reduction rate tended to be higher in lame ewes, which finally resulted in a significantly reduced DMY during the sixth ($P<0.05$, 1.78±0.034 kg and 1.63±0.063 kg of DMY for non-lame and lame ewes, respectively) and the seventh week of milking period ($P<0.01$, 1.76±0.029 kg and 1.58±0.055 kg of DMY for non-lame and lame ewes, respectively). The highest DMY reduction was observed during the 16^{th} week of milking period ($P<0.001$, 1.49±0.013 kg vs. 1.16±0.020 kg of DMY for non-lame and lame ewes, respectively). The reduction of DMY, for lame ewes, remained significant at $P<0.001$ level from the eighth to the 28^{th} week of milking. The reduction of DMY slowed down from the 29^{th} week of milking period ($P<0.01$, 0.83±0.034 kg and 0.65±0.063 kg of DMY for non-lame and lame ewes, respectively) to the 34^{th} week of milking period (end of lactation), when the differences were not significant (0.49±0.061 kg and 0.48±0.107 kg of DMY for non-lame and lame ewes, respectively).

Figure 2 shows the mean DMY of lame ewes and their controls per week, after adjusting for number and week of lactation, initiating from the fourth week before onset of lameness up to the eighth week afterwards. DMY tended to be lower for lame ewes, two weeks before lameness diagnosis (10.8%, $P=0.052$, 1.66 vs. 1.48 kg for control and lame ewes, respectively) and 16.1% lower one week before lameness diagnosis ($P\leq0.001$, 1.62 kg vs. 1.36 kg for control and lame ewes, respectively). At

285 the week of lameness diagnosis a significant milk yield reduction (P≤0.001) was
286 observed in lame ewes (about 32.5%), which was maximized one week later (35.8%,
287 P≤0.001). Figure 2 shows that for eight successive weeks after lameness diagnosis,
288 DMY of lame ewes continued to be significantly lower compared with controls at the
289 P≤0.001 level.

290 TMYs for non-lame and lame ewes, as calculated by the weekly solutions of
291 DMY produced by model 1, were 318.9 kg and 268.0 kg, respectively (a reduction of
292 about 16% for lame ewes). TMY, for the first 210 days of milking period, was 53.7 kg
293 lower (19.3%) in lame ewes compared with controls (P<0.01, Table 2) (Table 2 near
294 here).

295 **DISCUSSION**

296 This is a follow up study of a previously published work (Gelasakis at al.,
297 2010). In the aforementioned study, part of the data from Farm A was forced into a
298 different general linear model in order to calculate the effect of lameness on total milk
299 production; a reduction of about 20% was found. In the present study, a significant
300 decrease in lame ewes' DMY was observed when comparison was made both at flock
301 level on the prospective study and on individual ewe level (nested case-control study).
302 This is not surprising as a reduction on milk yield has also been reported in meat
303 sheep breeds (Winter, 2004) but research is limited.

304 Milk yield in dairy cows has been found to be lower (Rajala-Schultz *et al.*
305 1999; Warnick *et al.* 2001, Bicalho *et al.* 2008), equal (Martin *et al.* 1982) or even
306 higher (Dohoo and Martin, 1984) in cases of lameness. In lame, high yielding cows,
307 although a significant reduction in milk yield is expected, the latter remains at the
308 same or higher levels compared with herd average (Green *et al.* 2002), making rather

difficult the accurate assessment of the effect of lameness on milk yield (Huxley, 2013). This problem can be overcome by calculating lactation curves of lame cows and comparing them both with the average herd lactation curve and with those of appropriately selected controls (Barkema *et al.* 1994). A similar approach was used in our study.

The present study revealed that, DMY reduction started about four weeks before lameness diagnosis. The difference between lame ewes and their controls (Figure 2) became significant the week preceding diagnosis and continued for several weeks after recovery, which is in accordance with the long-term effect of lameness on milk yield in cows as reported by Green *et al.* (2002). This finding provides a reasonable explanation for the significant reduction both on DMY and on TMY observed in lame dairy ewes, even when the duration of lameness is short (less than a week). A loss of 50 kg of milk per lactation represents an income loss of about 45€ per case (mean price for sheep milk was 0.9€/kg during the 2013-2014 milking period in Greece). A treatment cost of 15-20€ per case must also be added. Obviously, depending on lameness incidence, losses can add up quickly and represent a significant financial burden for farmers.

Most cases of lameness in the present study were attributed to white line abscesses for which the aetiopathology remains unclear (Winter and Arsenos, 2009), although some evidence of genetic influences on the occurrence of white line lesions have recently been reported by Conington *et al.* (2010). Generally, increasing parity and herd size are considered probable risk factors of white line lesions (Barker *et al.* 2009); nutrition and other predisposing factors or stressors (for example, inadequate housing conditions) can have a direct effect on milk production but at herd or flock level, these are usually common to all animals. Farm-specific epidemiologic

investigation is needed to reveal differences related to management issues that pertain mainly to lame ewes. Moreover, the seasonal pattern of lameness occurrence within dairy sheep flocks needs to be further investigated and specified for the different causes of lameness. This could facilitate hypotheses making procedures regarding possible risk factors associated either with the productive cycle of dairy sheep or with the environmental conditions.

Is high milk production predisposing dairy ewes to lameness? The majority of cases in this study were diagnosed during the first four months postlambing, when milk yield was highest. However, due to the seasonal pattern of milk production the high lameness incidence coincided with the season that environmental humidity levels were also high (January to April). This is a major predisposing factor (Gelasakis *et al.* 2009) and may confound our results. Results reported in Table 2 imply that ewes selected as controls had higher milk production than the other non-lame ewes; they had an advantage of 53.7 kg of milk over lame ewes in the first 210 days of milking period whereas all non-lame ewes (controls included) had an advantage of 50.9 kg for the entire milking period (34 weeks). This is an indication that high milk production is indeed associated to lameness which is, also, supported by results from research on cows (Oikonomou *et al.* 2013).

The partial effects of different causes of lameness on milk yield were not possible to be estimated given the low number of cases per causative agent. Estimating the latter effects forms an interesting research topic for future studies on dairy sheep. However, the notion is that the negative effect of lameness on milk production could be due to the fact that stress and pain result in lower feed consumption. This is considered the major factor associated with decreased milk yield in meat sheep breeds, where, chronic lameness has been proved to have a significant

negative effect on body condition (Stewart *et al.* 1984; Marshall *et al.* 1991). Lame ewes may be underfed at pasture consuming a low quality and quantity of grass. This situation is certainly prevalent in animals covering their nutritional demands partially or totally from grazing. In our study, this scenario doesn't seem viable as the nutritional demands were covered by daily provision of an adequate ration in the shed. On Farm A, the grazing ground was very close to the shed and pasture quality was always very good. In any case, the highest prevalence of lameness and the subsequent reduction in milk yield were mainly observed during the winter months, when ewes didn't graze. A more reasonable hypothesis would be that lame ewes are not able to compete for a place at the feeding trough, which results on the consumption of lower quantity and, eventually, quality of feed. This scenario seems more viable in our case, even though feeding troughs provided, in theory, sufficient space for each ewe. In order to prove it, though, the behavioural pattern of intensively reared lame ewes should be assessed, using observational techniques, which forms an important subject of future research. Lower feed consumption could also result from the presence of inflammatory factors (e.g. cytokines interleukin-1 and interleukin-6); some of these factors are known to cause anorexia in laboratory animals (Harden *et al.* 2008).

The fact that milk production is already significantly lower one week prior to lameness diagnosis is a very promising observation. If it is confirmed in future studies it could become the basis for the development of an algorithm that could potentially warn farmers very early in the course of the disease. Similar approaches are already investigated in dairy cows (Machado *et al.* 2011; Van Hertem *et al.* 2013). Of course, automated milk recording is still an exception on dairy sheep farms but they are expected to become more common in the future. Early disease diagnosis is one potential way to justify the investment. In any case, though, the specificity of

monitoring milk yield as an early diagnostic tool for the identification of specific diseases needs to be assessed.

CONCLUSION

The results of this study demonstrate an evidence of significant financial losses in dairy sheep due to lameness, which though, need to be accurately estimated in further, more detailed, analyses. A large scale survey should now be considered in order to assess the effects of different causes of lameness, on different breeds of sheep, raised under different environments and management schemes.

ACKNOWLEDGEMENTS

The first author acknowledges financial support from the Greek State Scholarships Foundation (Athens, Greece). Participating farmers are acknowledged for their collaboration.

REFERENCES

Barkema, H.W., Westrik, J.D., van Keulen, K.A.S., Schukken, Y.H., Brand, A., 1994. The effects of lameness on reproductive performance, milk production and culling in Dutch Dairy Farms. Preventive Veterinary Medicine. 20, 249-259.

Barker, Z.E., Amory, J.R., Wright, J.L., Mason, S.A., Blowey, R.W., Green, L.E., 2009. Risk factors for increased rates of sole ulcers, white line disease, and digital dermatitis in dairy cattle from twenty-seven farms in England and Wales. Journal of Dairy Science. 92, 1971-1978.

405 Bicalho, R.C., Warnick, L.D., Guard, C.L., 2008. Strategies to analyze milk losses caused by
406 diseases with potential incidence throughout the lactation: a lameness example. Journal of
407 Dairy Science. 91, 2653-2661.

408 Boden, E., 1998. Black's Veterinary Dictionary. Ed. Adam & Charles Black, 10th Ed. London.

409 Conington, J., Nicoll, L., Mitchell, S., Bünger, L., 2010. Characterisation of white line
410 degeneration in sheep and evidence for genetic influences on its occurrence. Veterinary
411 Research Communications. 34, 481-489.

412 Coulon, J.B., Lescourret, F., Fonty, A., 1996. Effect of foot lesions on milk production by
413 dairy cows. Journal of Dairy Science. 79, 44-49.

414 De Rancourt, M., Fois, N., Lavín, M.P., Tchakérian, E., Vallerand, F., 2006. Mediterranean
415 sheep and goats production: An uncertain future. Small Ruminant Research. 62, 167-179.

416 Dohoo, I.R., Martin, S.W., 1984. Disease, production and culling in Holstein-Friesian cows.
417 IV. Effects of disease on production. Preventive Veterinary Medicine. 2, 755-770.

418 Espejo, L.A., Endres, M.I., Salfer, J.A., 2006. Prevalence of lameness in high-producing
419 Holstein cows housed in freestall barns in Minnesota. Journal of Dairy Science. 89, 3052-
420 3058.

421 Eze, C.A., 2002. Lameness and reproductive performance in small ruminants in Nsukka Area
422 of the Enugu State, Nigeria. Small Ruminant Research. 44, 263-267.

423 Fthenakis, G.C., El-masannat, E.T.S., Booth, J.M., Jones, J.E.T., 1991. Somatic cell counts of
424 ewes milk. British Veterinary Journal. 47, 575-581.

425 Gelasakis, A.I., Valergakis, G.E., Arsenos, G., 2009. Predisposing factors of sheep lameness.
426 Journal of the Hellenic Veterinary Medical Society. 59, 346-356.

427 Gelasakis, A.I., Arsenos, G., Valergakis, G.E., Fortomaris, P., Banos, G., 2010. Effect of
428 lameness on milk production in a flock of dairy sheep. The Veterinary Record. 167, 533-534.

Gelasakis, A.I., Valergakis, G.E., Arsenos, G., Banos, G., 2012. Description and typology of intensive Chios dairy sheep farms in Greece. Journal of Dairy Science. 95, 3070-3079.

Gelasakis, A.I., Oikonomou, G., Bicalho, R.C., Valergakis, G.E., Fthenakis, G.C., Arsenos, G., 2013. Clinical characteristics of lameness and potential risk factors in intensive and semi-intensive dairy sheep flocks in Greece. Journal of the Hellenic Veterinary Medical Society. 64, 123-130.

Green, L.E., Hedges, V.J., Schukken, Y.H., Blowey, R.W., Packington, A.J., 2002. The impact of clinical lameness on the milk yield of dairy cows. Journal of Dairy Science. 85, 2250-2256.

Green, L.E., George, T.R.N., 2008. Assessment of current knowledge of footrot in sheep with particular reference to Dichelobacter nodosus and implications for elimination or control strategies for sheep in Great Britain. The Veterinary Journal. 175, 173-178.

Harden, L.M., Du Plessis, I., Poole, S., Laburn, H.P., 2008. Interleukin (IL)-6 and (IL)-1β act synergistically within the brain to induce sickness behaviour and fever in rats. Brain, Behaviour and Immunity. 22, 838-849.

Hill, N.P., Murphy, P.E., Nelson, A.J., Mouttotou, N., Green, L.E., Morgan, K.L., 1997. Lameness and foot lesions in adult British dairy goats. The Veterinary Record. 141, 412-416.

Huxley, J.N., 2013. Impact of lameness and claw lesions in cows on health and production. Livestock Science. 156, 64-70.

Kaler, J., Green, L.E., 2008. Naming and recognition of six foot lesions of sheep using written and pictorial information: A study of 809 English sheep farmers. Preventive Veterinary Medicine. 83, 52-64.

Kaler, J., Wassink, G.J., Green, L.E., 2009. The inter- and intra-observer reliability of a locomotion scoring scale for sheep. The Veterinary Journal. 180, 189-194.

452 Leach, K.A., Whay, H.R., Maggs, C.M., Barker, Z.E., Paul, E.S., Bell, A.K., Main, D.,C.J.,
453 2010. Working towards a reduction in cattle lameness: 1. Understanding barriers to lameness
454 control on dairy farms. Research in Veterinary Science. 89, 311-317.

455 Machado, V.S., Caixeta, L.S., Bicalho, R.C., 2011. Use of data collected at cessation of
456 lactation to predict incidence of sole ulcers and white line disease during the subsequent
457 lactation in dairy cows. American Journal of Veterinary Research. 72, 1338-1343.

458 Marshall, D.J., Walker, R.I., Cullis, B.R., Luff, M.F., 1991. The effect of footrot on body
459 weight and wool growth of sheep. Australian Veterinary Journal. 68, 45-49.

460 Martin, S.W., Aziz, S.A., Sandals, W.C.D., Curtis, R.A., 1982. The association between
461 clinical disease, production and culling of Holstein-Friesian cows. Canadian Journal of
462 Animal Science. 62, 633-640.

463 Ministry of Agriculture Fisheries and Food, 1986. Manual of veterinary parasitological
464 laboratory techniques. HMSO, London.

465 Oikonomou, G., Cook, N.B., Bicalho, R.C., 2013. Sires predicted transmitting ability for
466 conformation and yield traits and previous lactation incidence of foot lesions as risk factors for
467 the incidence of foot lesions in Holstein cows. Journal of Dairy Science. 96, 3713-3722.

468 Phythian, C.J., Cripps, P.C., Grove-White, D., Jones, P.H., Michalopoulou, E., Duncan, J.S.,
469 2013. Observing lame sheep: evaluating test agreement between group-level and individual
470 animal methods of assessment. Animal Welfare. 22, 417-422.

471 Rajala-Schultz, P.J., Grohn, Y.T., McCulloch, C.E., 1999. Effects of Milk Fever, Ketosis, and
472 Lameness on Milk Yield in Dairy Cows. Journal of Dairy Science. 82, 288-294.

473 Russel, A.J.F., Doney, J.M., Gunn, R.G., 1969. Subjective assessment of fat in live sheep.
474 Journal of Agricultural Science. 72, 451-454.

Stewart, D.J., Clark, B.L., Jarrett, R.G., 1984. Difference between strains of Bacteroides nodosus in their effects on the severity of footrot, body weight and wool growth in Merino sheep. Australian Veterinary Journal. 61, 348-352.

Van Hertem, T., Maltz, E., Antler, A., Romanini, C.E.B., Viazzi, S., Bahr, C., Schlageter-Tello, A., Lockhorst, C., Berckmans, D., Halachmi, I., 2013. Lameness detection based on multivariate continuous sensing of milk yield, rumination, and neck activity. Journal of Dairy Science. 96, 4286-4298.

Warnick, L.D., Janssen, D., Guard, C.L., Gröhn, Y.T., 2001. The effect of lameness on milk production in dairy cows. Journal of Dairy Science. 84, 1988-1997.

Winter, A.C., 2004. Lameness in sheep. The Crowood Press, Ramsbury, Marlborough Wiltshire.

Winter, A.C., Arsenos, G., 2009. Diagnosis of white line lesions in sheep. In Practice. 31, 17-21.

FIGURE LEGENDS

Figure 1. Average lactation curves for i) non lame and ii) lame ewes (ewes diagnosed lame at least once across milking period) (95% confidence interval for the mean is used as measure of dispersion).

Figure 2. Mean DMY and lactation curves (adjusted for stage and number of lactation) of lame and control ewes for the period between four weeks before lameness diagnosis and eight weeks after it.